STATE OF MICHIGAN
IN THE SUPREME COURT

TERI WALTERS and KIM WALTERS,

Plaintiffs/Appellees,

-v-

S Ct No. 154489
COA No. 319016
LC No. 12-658-NH
Eaton County Circuit Court

DONALD S. FALIK d/b/a FALIK FAMILY DENTISTRY; DONALD S. FALIK, D.D.S.; ROBERT C. FALIK, D.D.S., and JANE DOE, jointly and severally,

Defendants/Appellants.
_____/

PLAINTIFFS-APPELLEES' SUPPLEMENTAL BRIEF RE LEAVE TO APPEAL (AFTER REMAND)
ON ORDER OF THE COURT DATED MAY 10, 2017

Respectfully submitted,

HERTZ SCHRAM PC
By: Daniel W. Rucker (P67832)
Attorneys for Plaintiffs/Appellees
1760 S. Telegraph Road, Suite 300
Bloomfield Hills, MI 48302
(248) 335-5000/Fax (248) 335-3346

SOMMERS SCHWARTZ PC
By: Jay G. Yasso (P45484)
Attorneys for Plaintiffs/Appellees
1 Towne Sq., Suite 1700
Southfield, MI 48076
(248) 335-0300/Fax (248) 936-1971

Dated: June 21, 2017

TABLE OF CONTENTS

INDEX OF AUTHORITIES

Cases

Statutes

Other Authorities

Rules

STATEMENT OF QUESTIONS INVOLVED
(AS ORDERED BY THE COURT

I. Whether the Court of Appeals erred in its interpretation of MCL 600.2955(1) and MRE 702?

The Circuit Court answers: "Yes"

Plaintiffs/Appellants answer: "No"

Defendants/Appellees answer: "Yes"

Court of Appeals answers: "No"

II. Whether the trial court erred in its application of those evidentiary standards or abused its discretion in granting the defendants' motions to exclude the plaintiff's testimony and for summary disposition?

The Circuit Court answers: "No"

Plaintiffs/Appellants answer: "Yes"

Defendants/Appellees answer: "No"

Court of Appeals answers: "Yes"

INTRODUCTION AND SUMMARY OF ARGUMENT

Defendants seek leave to review the Court of Appeal's reversal of an order granting Defendants' motion *in limine* to preclude the testimony of Plaintiffs' expert witness, Dr. M. Eric Gershwin. The Court of Appeals ("COA") determined that the trial court erroneously found Dr. Gershwin's testimony unreliable based on its interpretation of MRE 702 and MCL 600.2955(1) and its failure to consider information provided in support of the reliability of Dr. Gershwin's testimony. Dr. Gershwin is a preeminent expert in immunology, rheumatology, and autoimmune disease, which pertain to the autoimmune disease at issue, Wegener's granulomatosis ("WG"). The COA reversed the Circuit Court ("Circuit") order precluding Dr. Gershwin's testimony that Walters suffered the onset of WG as a result of her exposure to phosphoric acid etching solution.

WG is an extremely rare autoimmune disease that may be fatal and is anticipated to reduce Walters' lifespan by ten years. WG is a form of vasculitis that causes inflammation of the blood vessels, reduces blood flow to organs, and damages the kidneys, lungs, upper respiratory tract, and blood vessels, among other things. Walters suffered the onset of WG shortly after her exposure to a caustic etching solution made of phosphoric acid. Defendants erroneously provided this phosphoric acid etching solution to Walters instead of teeth whitening solution. The etching solution is used by dentists to destroy the enamel on teeth before bonding. Because of the highly caustic nature of the substance, it is used only by the dentist and is never sent home with a patient. Unfortunately, Walters used the etching solution in her dental tray overnight believing it to be teeth whitening gel as she requested. This led to severe injuries, including the onset of WG.

The parties provided dozens of scientific articles to the Circuit upon which Dr. Gershwin based his opinions. Dr. Gershwin relied upon the pathology of the disease, the time frame of its onset after exposure, and studies linking similar substances to WG onset. He traced the WG onset from effect to cause using a retrospective methodology found throughout the scientific

literature as the common practice because WG is not subject to experimental testing. Dr. Gershwin's analysis also satisfies the scientifically accepted SBH methodology, which examines nine factors to determine causation when experimental testing is not possible.

In its May 10, 2017 Order directing the Clerk to schedule oral argument on whether to grant the application for leave to appeal, the Court directed the parties to file supplemental briefs by June 21, 2017 on two issues. The two issues are listed below along with short answers to both.

Issue: "Whether the Court of Appeals erred in its interpretation of MCL 600.2955(1) and MRE 702?"

Short Answer: The COA properly rejected the Circuit's misapplication of MRE 702 and MCL 600.2955(1) upon *de novo* review and for abuse of discretion and found extensive support for Dr. Gershwin's reliability in the scientific literature and factual circumstances while acknowledging that he need not negate all possible causes or achieve absolute certainty. (COA Op. at 8-12 & n 7, **Ex. 50**.) The COA properly interpreted MCL 600.2955(1) and MRE 702 by focusing on Dr. Gershwin's principles and methodologies, rather than his ultimate conclusions; by properly abstaining from a search for absolute truth or uncontested evidence; by refraining from attempting to resolve a scientific dispute; and by examining whether Dr. Gershwin's opinion is rationally derived from a sound foundation. The COA found compelling an important list of considerations improperly ignored by the Circuit: (1) the caustic nature of phosphoric acid; (2) phosphoric acid being a WG-triggering environmental factor or chemical; (3) the intensity and duration of exposure; (4) the direct oral exposure in the presence of moisture; (5) the chronology of events, including the textbook timing of the immunological response from the date of exposure; (6) the incredible extent of the immune response; (7) the manifestation and duration of a classic WG symptom, sinusitis; (8) Walters' predisposition to WG; and (9) the support of scientific and medical literature either directly or by analogy. (COA Op. at 9.) The COA

correctly determined that this case is highly distinguishable from *Elher*, in which the expert based his opinion solely on his own personal beliefs. (COA Op. at 15.) Here, the reliability of Dr. Gershwin's principles and methodologies is supported by numerous factors such that his testimony was admissible and any challenge to the weight of his opinion is a matter for jury resolution. (COA Op. at 15.)

Issue: "Whether the trial court erred in its application of those evidentiary standards [MCL 600.2955(1) and MRE 702] or abused its discretion in granting the defendants' motions to exclude the plaintiff's testimony and for summary disposition?"

Short Answer: As a primary matter, the Walters point out that this matter arises from a motion in limine to preclude testimony, summary disposition was not requested, and the Circuit did not grant summary disposition. In its October 2, 2013 Order, the Circuit stated that it granted "Defendants' motion in limine to preclude the testimony of Dr. Gershwin for the reasons as stated on the record." (Order Granting Defs.' Mot. in Limine, Oct. 2, 2013, Cir. Ct. Dkt. No. 91, **Ex. 10**.) There is no discussion of summary disposition or dismissal in the underlying September 19, 2013 hearing transcript regarding the motion in limine. (Mot. *in Limine* Hrg. Tr., "Hrg. Tr.," Sept. 19, 2013, Cir. Ct. Dkt. No. 92, **Ex. 11**.) Teri Walters suffered damages other than the onset of WG, including injury to her mouth, teeth, gums, throat, airways, etc., and summary disposition of this ordinary negligence action would have been improper even if Dr. Gershwin's testimony is precluded regarding the causation of WG in Teri Walters.

As to preclusion of Dr. Gershwin's testimony, the Circuit committed both error and an abuse of discretion. The Circuit erred by requiring uncontested scientific opinions and establishing an overly rigorous admissibility standard demanding definitive proof of causation prior to trial, which is not contemplated under MRE 702, MCL 600.2955(1), or the relevant interpretive case law. The Circuit erred and abused its discretion by ignoring scientific literature, factual support,

and other considerations analyzed by the COA as well as by ignoring or misconstruing the following key components of Dr. Gershwin's opinions and methods. (i) Scientific articles establish connections between WG onset and environmental factors such as phosphorus, phosphates, fumes, solvents, and other chemicals sharing characteristics with phosphoric acid. (ii) Genetic predisposition is necessary for the onset of WG. (iii) Teri Walters' WG onset occurred subsequent to her exposure to phosphoric acid, and Dr. Gershwin testified her WG was a result of that exposure. (iv) The pathogenesis of WG is well documented in the scientific literature. (v) The scientific literature supports Dr. Gershwin's analysis of the role of cytoplasmic ANCA in WG onset and development. (vi) Causation of WG onset is supported in the scientific literature even though experimental testing is not feasible.

As a preeminent expert at the forefront of the scientific community for over forty years in the areas of immunology, rheumatology, and autoimmune disease, Dr. Gershwin's opinions, methods, and analysis of the published literature set the standard for sound opinions in the scientific community. The COA easily recognized the ample scientific support for Dr. Gershwin's opinions and that those opinions were rationally derived from a sound foundation, including commonly used scientific methods. The COA correctly reversed the Circuit's use of an overly rigorous gatekeeping standard, which demanded definitive proof with uncontested evidence, and the Circuit's abuse of discretion in ignoring the plentiful facts and literature confirming the reliability of Dr. Gershwin's scientific methodology and analysis.

STATEMENT OF FACTS

Plaintiffs-Appellees rely upon the Factual and Procedural Background set forth in their October 25, 2016 brief in response to Defendants' application for leave to appeal. Plaintiffs rely upon the exhibits and exhibit numbering in the prior brief and will refer to the exhibits in this brief consistent with the prior briefing.

ARGUMENT

The Circuit applied an inappropriate standard in interpreting and applying MRE 702 and MCL 600.2955(1) and improperly precluded Plaintiffs' expert witness from testifying on the life-shortening autoimmune disease, WG. The Circuit erred and abused its discretion by ignoring substantial scientific support for the expert's opinions on WG and the commonplace nature of his methods of scientific analysis. The COA properly reversed the Circuit as a matter of law for applying an incorrect standard and for abuse of discretion for ignoring facts supporting the expert's reliability. The COA easily distinguished *Elher*, involving expert opinion based only on person beliefs, from *Walters,* involving support from scientific literature, medical and chemical facts, and a chronology of phosphoric acid exposure, WG symptoms, and a textbook immune response. Leave to appeal should be denied. Alternatively, the Circuit's analysis should be rejected and the COA opinion affirmed. Dr. Gershwin's opinions are rationally derived from scientific articles and the application of accepted SBH and retrospective analysis methodologies.

APPLICABLE STANDARDS OF REVIEW

A trial court's interpretation of evidentiary rules or statutes affecting the admissibility of evidence is an issue of law subject to *de novo* review. *Chapin v A&L Parts, Inc*, 274 Mich App 122, 126; 732 NW2d 578 (2007), (citing *Waknin v Chamberlain*, 467 Mich 329, 332; 653 NW2d 176 (2002); *see also Mich DOT v Haggerty Corridor Partners Ltd P'ship*, 473 Mich 124, 133-134; 700 NW2d 380 (2005) (quoting *People v Lukity*, 460 Mich 484, 488; 596 NW2d 607 (1999) (holding that "whether a rule of evidence or statute precludes admissibility of the evidence" is a preliminary question of law subject to *de novo* review). A trial court's determination of whether to admit evidence is an issue subject to review for abuse of discretion. *Id*. A trial court's decision on a motion for reconsideration is also reviewed for an abuse of discretion. *Sherry v E Suburban Football League*, 292 Mich App 23, 31; 807 NW2d 859 (2011). An abuse of

discretion exists when the "decision results in an outcome falling outside the range of principled outcomes." *Id*. (quoting *Barnett v Hidalgo*, 478 Mich 151, 158; 732 NW2d 472 (2007).

In *Elher*, this Court did not challenge the COA application of *de novo* review to the trial court's use of appropriate principles in its gatekeeping role. *Elher v Misra*, 499 Mich 11; 878 NW2d 790 (2016). Instead, this Court found an *abuse of discretion by the trial court* in applying an inapplicable "testing" factor but determined that the *complete lack of support for any other reliability factors* required the exclusion of the witness. *Elher*, at 14-16 (**Ex. 51**). This Court examined the evidence and provided a detailed analysis of whether the trial court's gatekeeping effort resulted in a principled outcome. *Elher*, at 2-5, 12-16 (**Ex. 51**).

LEGAL PRECEDENT

Ordinary negligence requires (1) a legal duty, (2) breach, (3) damages, and (4) proximate cause. *Hill v Sears, Roebuck & Co*, 492 Mich 651, 660; 822 NW2d 190 (2012). Proximate cause for negligence is "well-settled" and requires both legal causation, or foreseeability, and cause-in-fact, or "but for" cause. *O'Neal v St John Hosp & Med Ctr*, 487 Mich 485, 496; 791 NW2d 853 (2010). Proximate cause is treated the same in medical malpractice and ordinary negligence cases. *Id*. There can be more than one proximate cause contributing to an injury, and all that is necessary is that the negligent act be "a proximate cause" of the injury rather than "the proximate cause." *Id*. at 496-97.

If there is evidence that points to any one theory of causation that indicates a "logical sequence of cause and effect," then there is a sufficient legal basis for that theory even if there exist "other plausible theories" of causation also with support in the evidence. *Skinner v Square D Co*, 445 Mich 153, 164; 516 NW2d 475 (1994) (quotation omitted). A plaintiff's burden of causation is to "present substantial evidence from which a jury may conclude that *more likely than not*, but for the defendant's conduct, the plaintiff's injuries would not have occurred." *Id*. at 164-65 (emphasis added). The burden is satisfied by "specific facts that would support a reasonable inference of a logical sequence of cause and effect." *Id*. at 174. Where multiple factors produce an injury, factual

causation is satisfied where the "defendant's actions, *more likely than not*, were *a 'substantial factor'* in producing a plaintiff's injuries." *Id*. at 165 n8 (citation omitted, emphasis added). An evidentiary record may not be submitted to the jury that allows the "jury to do nothing more than guess." *Id*. at 174. Instead, causation is satisfied by evidence showing "a reasonable likelihood of probability rather than a possibility," but the evidence "need not negate all other possible causes." *Id*. at 166 (quoting 57A Am Jur 2d, Negligence, § 461, p 442). "Absolute certainty" is not required or possible when proving negligence by circumstantial evidence, and causation is sufficiently established by a "chain of circumstances" resulting in a "conclusion which is more probable than any other hypothesis reflected by the evidence." *Id*.

"The critical inquiry with regard to expert testimony is whether such testimony will aid the factfinder in making the ultimate decision in the case." *People v Coy*, 243 Mich App 283, 294-295; 620 NW2d 888 (2000). MRE 702 permits expert testimony under the following circumstances:

> If the court determines that scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education may testify thereto in the form of an opinion or otherwise if (1) the testimony is based on sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case. (MRE 702.)

In addition, the provisions of MCL 600.2955 should inform this Court's analysis of whether expert opinion is appropriate. That statutory section states in relevant part that,

> In an action for the death of a person or for injury to a person or property, a scientific opinion rendered by an otherwise qualified expert is not admissible unless the court determines that the opinion is reliable and will assist the trier of fact. In making that determination, the court shall examine the opinion and the basis for the opinion, which basis includes the facts, technique, methodology, and reasoning relied on by the expert, and shall consider all of the following factors:
>
> (a) Whether the opinion and its basis have been subjected to scientific testing and replication.
>
> (b) Whether the opinion and its basis have been subjected to peer review publication.
>
> (c) The existence and maintenance of generally accepted standards governing the application and interpretation of a methodology or technique and whether the opinion and its basis are consistent with those standards.
>
> (d) The known or potential error rate of the opinion and its basis.
>
> (e) The degree to which the opinion and its basis are generally accepted within the

> relevant expert community. As used in this subdivision, "relevant expert community" means individuals who are knowledgeable in the field of study and are gainfully employed applying that knowledge on the free market.
>
> (f) Whether the basis for the opinion is reliable and whether experts in that field would rely on the same basis to reach the type of opinion being proffered.
>
> (g) Whether the opinion or methodology is relied upon by experts outside of the context of litigation. (MCL § 600.2955(1).)[1]

In evaluating the reliability of expert opinion, "the inquiry is flexible and focused 'solely on principles and methodology' rather than ultimate conclusions." *Chapin*, 274 Mich App at 127 (quoting *Daubert v Merrell Dow Pharmaceuticals, Inc*, 509 US 579, 594-95 (1993)). A court's "role as gatekeeper does not require it to search for absolute truth, to admit only uncontested evidence, or to resolve genuine scientific disputes" even where there is conflicting evidence or opinions. *Id*. In the case of conflicts of evidence or opinion, the expert opinion is admissible "as long as the opinion is rationally derived from a sound foundation." *Id*. Section 600.2955(1) "does not require that each and every one of those seven factors must favor the proffered testimony." *Id*. at 137; *see also Elher*, at 13 and n 23, 14-15 (**Ex. 51**) (application of improper factors may be an abuse of discretion). "The standard focuses on the scientific validity of the expert's methods rather than on the correctness or soundness of the expert's particular proposed testimony." *People v Unger*, 278 Mich App 210, 217-18, 220; 749 NW2d 272 (2008). This distinction between reliable methods and debatable expert opinions is important because "not every particular factual circumstance can be the subject of peer-reviewed writing," and when unique facts arise, and there is not medical or scientific literature in support of an expert's conclusions, it is up to counsel to cross-examine the experts and it is up to the jury to determine which expert is

---

[1] The trial court never mentioned MCL 600.2955(2). Instead, the judge expressly stated that "I'm considering the rules set forth in MRE 702, the statute 600.2955(1) and the case law." (Hrg. Tr. at 19, **Ex. 11**.) MCL 600.2955(2) is not applicable. There is no "novel methodology or form of scientific evidence" at issue. Dr. Gershwin used the same method of tracking cause and effect as is shown in decades of peer-reviewed case control WG studies. (*See, e.g.*, **Exs. 18, 20, 21, 25, 26**.)

more credible. *Id*. at 220. Where conflicting opinions of experts arise, it is a matter of credibility for the jury to resolve. *See Martin v Ledingham*, 488 Mich 987, 987-88; 791 NW2d 122 (2010) (contrary expert opinions regarding standard of care created a jury question).[2] The "gatekeeper role applies to all stages of expert analysis" and "mandates a searching inquiry" of the underlying data and the expert interpretation and extrapolation of the data. *Gilbert v DaimlerChrysler Corp*, 470 Mich 749, 782; 685 NW2d 391 (2004).

In evaluating the MCL 600.2955 factors, an abuse of discretion occurs when a court applies an inapplicable factor, such as "scientific testing and replication" in *Elher*, but a *complete lack of support for any other reliability factors* requires exclusion of an expert witness despite an abuse of discretion as to another factor. *Elher*, at 14-16 (**Ex. 51**). The *Daubert* factors may or may not be relevant in assessing reliability, depending on the nature of the issue, the expert's expertise, and the subject of the expert's testimony. *Elher*, at 13 (**Ex. 51**) (citing *Kumho Tire Co v Carmichael*, 526 US 137, 150 (1999)). A court does not necessarily abuse its discretion even in relying only on two of the MCL 600.2955 factors. *Elher*, at 13-14 (**Ex. 51**). An expert's reliance on his own "belief system" or "his own beliefs" without "any supporting authority," with "no medical literature supporting his opinion," in the face of contradictory expert opinion and contradictory published literature, without showing "some basis in fact" for the opinion, and without "any other support for [the expert's] opinion," is insufficient to establish reliability and

---

[2] Defendants have cited an affidavit of Dr. Monika Mohan, who affirmed under oath that, "***within a reasonable degree of medical certainty***," Mrs. Walters' use of phosphoric acid etching solution "was not the cause of her" WG. (Defs.' Leave App., Ex. C, Aff. of Dr. Mohan ¶ 3, emphasis added.) ***Defendants contend that experts do have the ability to determine with medical certainty whether or not a particular factor caused WG in Teri Walters***. Notably, Dr. Mohan's affidavit provides no "inconsistent" or "contradictory" hypotheses of causation, let alone one that is "*equally*" supported by the evidence as is required by *Skinner*, 445 Mich at 166-67, so there is nothing to weigh against Dr. Gershwin's analysis at this point. Moreover, a conflict of expert opinions should be left for jury resolution. "The courts are not in the business of resolving scientific disputes." *Chapin*, 274 Mich App at 139. "The courts are unlikely to be capable of achieving a degree of scientific knowledge that scientists cannot." *Id*.

admissibility under MCL 600.2955 and MRE 702. *Elher* at 8, 15-16. While "peer-reviewed, published literature is not always necessary or sufficient," an expert opinion may fail these admissibility standards based upon "the lack of supporting literature, combined with the lack of any other form of support," particularly where "there is contradictory medical literature." *Id*. at 16, also at 3 ("did not provide any supporting authority for his opinion"). A lack of supporting literature is "not dispositive" but it is "an important factor" in determining admissibility of expert testimony. *Id*. at 11. MRE 702 "generally" requires more than evidence of an expert's background and experience to establish a reliable and admissible opinion. *Id*. MCL 600.2955(2) is not applicable based upon the presence of a novel *opinion*, but it applies only to a novel *methodology or form of scientific evidence*. *Id*. at 14 n27.

In a motion for reconsideration, "[t]he moving party must demonstrate a palpable error by which the court and the parties have been misled and show that a different disposition of the motion must result from correction of the error." MCR 2.119(F)(3).

I. The Court of Appeals Did Not Err In Its Interpretation Of MCL 600.2955(1) And MRE 702.

The Court of Appeals did not err in interpreting MCL 600.2955(1) and MRE 702. Plaintiffs set forth *infra* various holdings and analyses of the COA that meet the requirements of MCL 600.2955(1) and MRE 702 and the case law setting out the standards for interpreting those guidelines. Plaintiffs rely on the factual information and exhibits from their prior briefing, filed with this Court on October 25, 2016. Discussed below, the COA found nine factors supported the reliability of Dr. Gershwin's opinion under MRE 702 and MCL 600.2955(1). (COA Op. at 9.)

(1) The caustic nature of phosphoric acid. (COA Op. at 9.)

The COA referred to the Ultradent phosphoric acid "Safety Data Sheet" and its indication of a variety of harmful characteristics, including dangers of "permanent tissue damage," "***corrosive***,

causes skin burning," "harmful if swallowed," "***irritating to respiratory system***," and by listing as ***"conditions to avoid" the "exposure to moist air or water."*** (COA Op. at 9; Safety Data Sheet at 1-2, **Ex. 22**; Pls' 10-25-16 MSC Br. at 25, 27.) All these characteristics are relevant to the onset of WG, which Dr. Gershwin and the scientific community, established through peer-reviewed articles, agree is initiated through the *inflammation of the patient's airways*. (*See* Pls' 10-25-16 MSC Br. at 24-27.) Plaintiffs cited at least five peer-reviewed articles confirming the common scientific understanding that WG onsets by inflammation of the airways. (*See* Pls' 10-25-16 MSC Br. at 24-26, citing Hamidou, **Ex. 19** at 373-74; Mahr, **Ex. 18** at S-87; Duna, **Ex. 26** at 669-70, 673; Chen, **Ex. 23** at A293, A296 and Fig. 1; and Sibelius, **Ex. 24** at 497-98 articles; COA Op. at 3-4 (citing all but Sibelius).) The peer-reviewed literature states that WG is triggered by inflammation of the airways. Dr. Gershwin testified that WG begins with acute inflammation of the upper airways, phosphoric acid produces incredible inflammation, and his opinion is that the phosphoric acid caused the inflammation necessary in Teri Walters. (*See* Pls' 10-25-16 MSC Br. at 24-25; Dr. Gershwin Tr. at 12-14, 29, 33, **Ex. 9**.) As discussed *infra*, Teri Walters was diagnosed first with a respiratory disease, sinusitis, followed by the diagnosis of WG. Dr. Gershwin testified, with support from peer-reviewed literature, that sinusitis is a first phase symptom of WG. (*See* Pls' 10-25-16 MSC Br. at 24 (citing Hamidou, **Ex. 19**) and at 36 (citing Dr. Gershwin testimony) and at 4-5 (multiple diagnoses of sinusitis followed by WG diagnosis).)

The COA's reliance on the caustic nature and characteristics of the Ultradent phosphoric acid given to Teri Walters by Defendants helps a jury to understand the evidence or determine a fact in issue. MRE 702. Dr. Gershwin can explain to the jury, with support from the scientific community, that phosphoric acid has the caustic and inflammatory characteristics necessary to cause respiratory inflammation, which is the first step in the onset of WG. The Safety Data Sheet provides appropriate facts or data upon which to establish the chemical's caustic nature and

capacity to inflame the airways. MRE 702(1). At least five peer-reviewed articles support the reliability of Dr. Gershwin's opinion and methods of searching for a caustic and inflammatory chemical linked to the onset of WG in Teri Walters. MRE 702(2). Dr. Gershwin's analysis that the Ultradent phosphoric acid carries the capacity to cause inflammation of the airways is a reliable application of the facts in this case, including the caustic and inflammatory characteristics set forth in the Safety Data Sheet, the fact of Teri Walters' use of the acid in her teeth-whitening dental trays, and the onset of WG in Teri Walters starting from respiratory inflammation initially diagnosed as sinusitis. MRE 702(3).

The COA properly considered that peer reviewed articles support Dr. Gershwin's opinion and basis regarding a caustic and inflammatory substance having the capacity to initiate WG through inflammation of the airways. MCL 600.2955(1)(b). Particularly with respect to the Sibelius article describing the pathology of WG onset starting with respiratory "burst" or inflammation, but also with respect to at least the Hamidou, Mahr, Duna, and Chen articles confirming the onset of WG from respiratory inflammation, there is general acceptance of Dr. Gershwin's opinion, basis, and methodology with respect to linking a caustic and inflammatory substance, phosphoric acid, to the onset of WG. MCL 600.2955(1)(c)(e). At least these five articles establish that experts outside of litigation would rely upon and seek out the presence of and exposure to a caustic and inflammatory substance generating the respiratory inflammation leading to WG onset. MCL 600.2955(1)(f)(g).

The COA's opinion as to the caustic nature of the phosphoric acid comports with the case law interpreting admissibility of expert testimony. The COA properly examined Dr. Gershwin's method (Safety Data Sheet, medical records, and at least five articles regarding WG onset through airway inflammation) of establishing phosphoric acid as a caustic and inflammatory substance capable of creating the respiratory inflammation linked to the onset of WG rather than

dwelling upon Dr. Gershwin's ultimate conclusion. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95) (focus on principles and methods rather than conclusions). The COA did not search for absolute truth, require only uncontested evidence, or try to resolve a scientific dispute in analyzing the link between the caustic and inflammatory nature of phosphoric acid and the inflammation precipitating WG onset. *Id*.; *also Unger*, 278 Mich App at 217-18, 220. The COA properly relied upon the nature of phosphoric acid, as established by the Safety Data Sheet and testimony, and the recognition that WG onsets from respiratory inflammation, as established in multiple peer-reviewed articles, as a sound foundation for the rationally derived opinion of Dr. Gershwin that the phosphoric acid triggered inflammation in Teri Walters that onset WG. *Chapin*, 274 Mich App at 127. There was no conflicting medical opinion presented by any defense expert regarding the nature of phosphoric acid or the role of inflammation in WG onset, but even if there had been, it would have been up to the jury to resolve such credibility issues of experts. *Unger*, 278 Mich App at 220; *Martin*, 488 Mich at 987-88. The opinion of Dr. Gershwin regarding the caustic and inflammatory nature of phosphoric acid relative to the onset of WG through inflammation of the airways was not a matter of his "own beliefs," it was supported by documentation and scientific literature, and there was presented no contradictory expert opinion or published literature on the matter. *Elher*, at 3, 8, 15-16. Thus, the COA's reliance on the nature of the phosphoric acid in combination with the recognized inflammation trigger for WG onset was consistent with MRE 702, MCL 600.2955(1), and the interpretive case law.

(2) Phosphoric acid is a WG-triggering environmental factor or chemical. (COA Op. at 9-10.)

The COA referred to phosphoric acid as being a WG-triggering environmental factor or chemical and analyzed the scientific literature presented stating that while a "definitive" or

"absolutely certain" etiology for WG is unclear or unknown, the articles reflect confirmation by the scientific community that WG onset "probably" involves genetic susceptibility combined with environmental factors, and "current understanding" recognizes the interplay of genetic and environmental risk factors, studies are "consistent in finding positive associations" between environmental factors and WG risk, environmental factors are "important" in the development of ANCA associated with WG onset, and that WG includes the "predominant involvement of the airways" leading to the conclusion that an inhaled agent may trigger WG onset. (COA Op. at 9-10, citing articles by Hamidou, **Ex. 19**; Mahr, **Ex. 18**; Chen, **Ex. 23**; Duna, **Ex. 26**.) In the Duna article and another cited by the COA, researchers indicated statistically significant association between environmental factors such as pesticides, insecticides, fumes, particulates, and WG onset. (COA Op. at 4-5, 10, citing Lane, **Ex. 25** at 814-15, 820; Duna, **Ex. 26** at 669; *also* Lane, **Ex. 20** at 272 (statistically significant association with farming.) The Walters provided the COA with numerous articles addressing environmental factors associated with WG onset. (Pls' 10-25-16 MSC Br. at 19-20, 22-23, 25-26, 28-29.) Dr. Gershwin analyzed data and studies regarding the onset of WG caused by a variety of environmental triggers and applied that information by analogy to Teri Walters' exposure to a much more potent chemical. (Aff. of Dr. Gershwin ¶ 2, **Ex. 17**.) He conducted a retrospective review of Teri Waters' condition from the point of the display of symptoms to a period several weeks before, as if one has found a footprint and is tracing the footprints back to their origin. (*See, e.g.*, Dr. Gershwin Tr. at 27:9 - 28:11, 32:19-22, 33:14-25, **Ex. 9**.) The scientific articles fully justify the COA's reliance upon the importance of environmental factors in WG onset and supporting the reliability of Dr. Gershwin's methods and opinion tracing back from symptoms to an environmental factor, phosphoric acid, which caused the onset of WG in Teri Walters.

Testimony that environmental factors are involved in the onset of WG aids a jury in

understanding the relation of exposure to phosphoric acid to the onset of WG in Teri Walters. MRE 702. Dr. Gershwin's testimony regarding exposure to an environmental factor is based upon sufficient facts or data because the evidence shows that Teri Walters was exposed to phosphoric acid given to her by Defendants and Teri Walters suffered the onset of WG. MRE 702(1). (Pls' 10-25-16 MSC Br. at 3-5.) Numerous peer-reviewed articles provided by the Walters and/or relied upon by the COA support the reliability of Dr. Gershwin's opinion and methods of searching for an environmental factor that initiated the onset of WG in Teri Walters. MRE 702(2). Dr. Gershwin reliably applied his methods to the present facts by seeking out an environmental factor to which Teri Walters was exposed in the appropriate time frame prior to the presentation of WG symptoms (see discussion *infra*), the environmental factor Dr. Gershwin discovered has caustic and inflammatory characteristics consistent with the inflammation of the airways involved in WG onset (see discussion *supra*), Teri Walters was exposed to an environmental factor, phosphoric acid, in the appropriate time frame and with the characteristics necessary to initiate WG, and the phosphoric acid shares characteristics with, but is far more potent than, a wide variety of other chemicals associated with or statistically significantly associated with the onset of WG (discussed *supra* and *infra*). MRE 702(3).

The COA again considered that peer reviewed articles support Dr. Gershwin's opinion and basis regarding the involvement of environmental factors in WG onset. MCL 600.2955(1)(b). The articles presented to and reviewed by the COA uniformly establish a general acceptance of Dr. Gershwin's opinion, basis, and methodology with respect to linking an environmental factor to the onset of WG. MCL 600.2955(1)(c)(e). The articles demonstrate that experts outside of litigation would rely upon and seek out the presence of an environmental factor triggering WG onset. MCL 600.2955(1)(f)(g).

The COA's opinion regarding the importance of an environmental factor in WG onset

comports with the case law interpreting admissibility of expert testimony. The COA properly examined Dr. Gershwin's method of seeking out an environmental factor to which Teri Walters was sufficiently exposed, in the right time frame relative to the immunological response, capable of the necessary inflammation of the airways, and bearing the potency and characteristics of other substances associated with or significantly associated with the onset of WG in scientific literature, and the COA properly refrained from challenging Dr. Gershwin's ultimate conclusion. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95) (focus on principles and methods rather than conclusions). The COA did not search for absolute truth, require only uncontested evidence, or try to resolve a scientific dispute in analyzing the link between environmental factors and WG onset. *Id*.; *also Unger*, 278 Mich App at 217-18, 220. The COA properly relied upon evidence of Teri Walters intense and lengthy exposure to phosphoric acid, the time frame of exposure relative to WG symptom onset, the shared characteristics and greater potency of phosphoric acid relative to other environmental factors associated with or significantly associated with WG onset as a sound foundation for the rationally derived opinion of Dr. Gershwin that the phosphoric acid is an environment factor that triggered the onset of WG in Teri Walters. *Chapin*, 274 Mich App at 127. There was no conflicting medical opinion presented by any defense expert regarding any other environmental factor involved in Walters' WG onset, but even if there had been, it would have been up to the jury to resolve such credibility issues of experts. *Unger*, 278 Mich App at 220; *Martin*, 488 Mich at 987-88. The opinion of Dr. Gershwin regarding an environmental factor in the onset of WG not a matter of his "own beliefs," it was supported by documentation and scientific literature, and there was presented no contradictory expert opinion or published literature on the matter. *Elher*, at 3, 8, 15-16. Thus, the COA's reliance on Dr. Gershwin's identification of an environmental factor sufficient to trigger WG onset was consistent with MRE 702, MCL 600.2955(1), and the

interpretive case law.

(3) The intensity and duration of exposure. (COA Op. at 9.)

The COA determined that there was no dispute as to the intensity and duration of Teri Walters' exposure to phosphoric acid because she slept with it in her dental trays overnight. (COA Op. at 9.) Teri Walters testified that after sleeping with the phosphoric acid solution in her mouth all night, she awoke with burning sensations inside her mouth, including along her gumline and the sides of her tongue. (TW Tr. at 43:3-25, 48:10-17, **Ex. 1**.) Dr. Robert Falik testified that the solution etches the teeth, and a little drop is left on the tooth for about 20 seconds only. (RF Tr. at 26:3-7, **Ex. 5**.) Dr. Donald Falik agreed that etching solution should only remain on the tooth for about 20 seconds and that the etching solution dissolves mineral from the tooth enamel and creates "literally miles of microscopic fingerlets" in the tooth. (DF Tr. at 45:20-46:10, **Ex. 4**.) Dr. Robert Falik also testified that etching solution can destroy a tooth if left on for a long period of time. (RF Tr. at 26:11-14, **Ex. 5**; *see also* Resp. 1st Interrogs.-Admit., Admit 8, **Ex. 3**.) Dr. Gershwin testified that the high level of ANCA and the acute onset of WG in Walters from intense exposure to phosphoric acid were consistent with studies of silica exposure showing that the intensity of exposure, rather than duration of exposure, is more important in initiating WG. (Dr. Gershwin Tr. at 26:2-11, 28:5-11, **Ex. 9**.) (*See* Mahr, **Ex. 18** at S-87 (intensity of exposure is associated with WG onset more so than duration of exposure); *also* Lane, **Ex. 25** at 820 (addressing silica and indicating that "most previous studies" indicate that duration of exposure is less important than intensity of exposure); Duna, **Ex. 26** at abstract (acknowledging that "intensity of exposure" to inhaled potential precipitants of WG may have varied without detection by the study).) As discussed *supra*, at least five peer-reviewed articles confirm the common scientific understanding that WG onsets by inflammation of the airways. (*See* Pls' 10-25-16 MSC Br. at 24-26, citing Hamidou, Mahr, Duna, Chen, and Sibelius articles; COA Op. at

3-4 (citing all but Sibelius).) These articles regarding intensity and duration of exposure, the key factor of inflammation of the airways, and the uncontested nature of Teri Walters' intense and lengthy exposure to phosphoric acid support the COA finding reliable Dr. Gershwin's method of analysis and opinion that phosphoric acid triggered WG in Teri Walters.

Expert testimony regarding intensity and duration of exposure to an environmental factor involved in the onset of WG aids a jury in understanding the relevance and weight of the fact that Teri Walters slept with phosphoric acid in her mouth just before the onset of WG symptoms. MRE 702. Dr. Gershwin's testimony regarding duration and intensity of exposure is based upon sufficient facts or data because the evidence shows that Teri Walters slept with the phosphoric acid in her dental trays. MRE 702(1). (Pls' 10-25-16 MSC Br. at 3-4.) Peer-reviewed articles addressing intensity and duration of exposure as well as the importance of inflammation of the airways to the onset of WG support the reliability of Dr. Gershwin's opinion and methods based upon Teri Walters' lengthy and intense exposure to phosphoric acid. MRE 702(2). Dr. Gershwin reliably applied his method of seeking a lengthy and intense exposure, as the facts are undisputed that Teri Walters slept with the phosphoric acid in her mouth even though such a chemical should only be on the tooth for about 20 seconds to remove the enamel and the substance is otherwise potent enough to destroy teeth. MRE 702(3).

Peer reviewed articles support Dr. Gershwin's opinion and basis regarding the import of duration and intensity of exposure to an inflammatory substance. MCL 600.2955(1)(b). The articles presented to and reviewed by the COA suggest a general acceptance that the onset of WG is associated with the duration and intensity of exposure to an environmental factor. MCL 600.2955(1)(c)(e). The articles show experts outside of litigation would rely upon and examine the duration and intensity of exposure to a substance prior to WG onset. MCL 600.2955(1)(f)(g).

The COA's opinion regarding the importance of duration and intensity of exposure in WG

onset comports with the case law interpreting admissibility of expert testimony. The COA properly considered Dr. Gershwin's evaluation of the intensity and duration of exposure to phosphoric acid without improperly challenging Dr. Gershwin's ultimate conclusion. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95) (focus on principles and methods rather than conclusions). The COA did not search for absolute truth, require only uncontested evidence, or try to resolve a scientific dispute in analyzing the link between intensity and duration of exposure and WG onset. *Id*.; *also Unger*, 278 Mich App at 217-18, 220. The COA properly relied upon undisputed evidence of Teri Walters intense and lengthy exposure to phosphoric acid as providing a sound foundation for the rationally derived opinion of Dr. Gershwin that the intense and lengthy exposure to phosphoric acid triggered the onset of WG. *Chapin*, 274 Mich App at 127. There was no conflicting medical opinion presented by any defense expert regarding the impact of intensity and duration of exposure, but even if there had been, it would have been up to the jury to resolve such credibility issues of experts. *Unger*, 278 Mich App at 220; *Martin*, 488 Mich at 987-88. The opinion of Dr. Gershwin regarding intensity and duration of exposure is not a matter of his "own beliefs," it was supported by documentation and scientific literature, and there was presented no contradictory expert opinion or published literature on the matter. *Elher*, at 3, 8, 15-16. Thus, the COA's reliance on Dr. Gershwin's consideration of intensity and duration of exposure was consistent with MRE 702, MCL 600.2955(1), and the interpretive case law.

(4) The direct oral exposure in the presence of moisture. (COA Op. at 9.)

The COA found Dr. Gershwin's consideration of Teri Walters' direct oral exposure to phosphoric acid as another factor in support of the reliability of his methods and reasoning. The COA properly relied upon this consideration for the reasons set out in the discussions of the importance of inflammation of the airways and of intensity and duration of exposure, *supra*. The

fact that Teri Walters slept with the phosphoric acid in her mouth overnight factors into both the inflammation analysis and the intensity analysis. The COA correctly recognized that there was no dispute that Teri Walters slept with the phosphoric acid in her dental trays. (COA Op. at 9.)

(5) The chronology of events, including the textbook timing of the immunological response from the date of exposure. (COA Op. at 9.)

The COA correctly acknowledged that the timing of WG symptom onset relative to her exposure to phosphoric acid was a textbook time frame for an immunological response. (COA Op. at 9; *see, e.g.*, Dr. Gershwin Tr. at 26:23 - 27:8, **Ex. 9**.) There was no dispute of this issue, as Dr. Gershwin has written, among other things, textbooks on sinus disease that address WG. Gershwin, M.E., *et al*., *Diseases of the Sinuses, A Comprehensive Textbook of Diagnosis and Treatment* (Humana Press, 1996, 2013) (cover page, **Ex. 15**).

Expert testimony regarding the timing of an immunological response compared to the medically anticipated immunological response time between cause and effect is important in assisting a jury in understanding the relevant time period in which Teri Walters may have been exposed to an environmental factor that caused the onset of WG symptoms. MRE 702. The facts and data upon which Dr. Gershwin relied come from Teri Walters' specific testimony and affidavit statement regarding her use of the phosphoric acid on February 11, 2011, the night before her son's military ball, followed by diagnoses of symptoms of WG, particularly sinusitis, and then WG with the medical records indicating about three to four weeks between use of the phosphoric acid and Teri Walters' observation of symptoms. MRE 702(1). (Pls' 10-25-16 MSC Br. at 3-5 and nn1-2.) This issue was not challenged, but Dr. Gershwin's familiarity with the textbook time frames, having helped write the textbooks, supports the reliability of Dr. Gershwin's opinion and methods. MRE 702(2). Dr. Gershwin reliably applied his method of evaluating the textbook time frame for immunological response by examining the date of Teri

Walters' exposure compared to the medical records' indication of the time frame for onset of symptoms. MRE 702(3).

The unchallenged textbook was peer reviewed by the co-editor. MCL 600.2955(1)(b). The uncontested and textbook nature of the time frame of immunological response suggests general acceptance. MCL 600.2955(1)(c)(e). The textbook immunological response time frame is something experts outside of litigation would rely upon. MCL 600.2955(1)(f)(g).

The COA's opinion regarding the time frame between exposure and WG symptom onset comports with the case law interpreting admissibility of expert testimony. The COA properly considered Dr. Gershwin's evaluation of the textbook time frame without improperly challenging Dr. Gershwin's ultimate conclusion. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95) (focus on principles and methods rather than conclusions). The COA did not search for absolute truth, require only uncontested evidence, or try to resolve a scientific dispute in analyzing the time frame between exposure and symptom onset. *Id*.; *also Unger*, 278 Mich App at 217-18, 220. The COA properly relied upon undisputed evidence of the time frame between exposure and symptom onset as well as the unchallenged nature of Dr. Gershwin's textbook information as providing a sound foundation for the rationally derived opinion of Dr. Gershwin that the onset of WG could be traced back temporally to Teri Walters' exposure to phosphoric acid. *Chapin*, 274 Mich App at 127. There was no conflicting expert opinion presented suggesting that the immunological response time was something different, but even if there had been, it would have been up to the jury to resolve such credibility issues of experts. *Unger*, 278 Mich App at 220; *Martin*, 488 Mich at 987-88. The opinion of Dr. Gershwin regarding the comparison of immunologic response and the time frame exposure and symptom onset is not a matter of his "own beliefs," it was unchallenged textbook information, and there was presented no contradictory expert opinion or published literature on the matter. *Elher*, at 3, 8, 15-16. Thus,

the COA's reliance on Dr. Gershwin's consideration of the time frame between exposure and symptom onset was consistent with MRE 702, MCL 600.2955(1), and the interpretive case law.

(6) The incredible extent of the immune response. (COA Op. at 9.)

The COA recognized the incredible extent of the immunological response in Teri Walters marked by a high-titer ANCA. (COA Op. at 9; *see, e.g.*, Dr. Gershwin Tr. at 26:23 - 27:8, **Ex. 9**.) Dr. Gershwin testified that the phosphoric acid etching solution caused the autoimmune effects in Walters because of her genetic predisposition to WG, which was exhibited by an acute presentation of an abundance of ANCA. (Dr. Gershwin Tr. at 26:3-11, 35:7-10, **Ex. 9**.) Dr. Gershwin explained that a high level of the antibody ANCA (antineutrophil cytoplasmic antibody) is indicative of WG. (*See, e.g.*, Dr. Gershwin Tr. at 15:5-6, 16:12-13, **Ex. 9**.) Dr. Gershwin specified the *cytoplasmic* antigen inducing the ANCA related to WG. (Dr. Gershwin Tr. at 15:19 – 16:10, **Ex. 9**.) There was no dispute as to the presence of an abundance of ANCA in Teri Walters. (COA Op. at 9.) The scientific literature establishes the presence of high levels of ANCA in WG onset. (Chen, **Ex. 23** at A293, A296 and Fig. 1 ("ANCA are serological hallmarks" for small vessel vasculitis such as WG; environmental factors stimulate inflammation, generating expression of neutrophil adhesion molecules and movement of ANCA antigens to the cell surfaces, resulting in the binding of ANCA with antigens on the cell surfaces, leading to the adherence of the neutrophils to blood vessel walls); Sibelius, **Ex. 24** at 497, 502 (ANCAs are "anti-neutrophil cytoplasmic antibodies"; cytoplasmic ANCA (c-ANCA) is a seromarker of WG that is 95 percent specific to WG and the higher titer of c-ANCA correlates with higher activity level of WG because c-ANCA functions in the pathogenesis of WG).

Expert testimony regarding the role of c-ANCA in the onset and activity of WG and the import of high levels of c-ANCA in Teri Walters aids the jury in understanding that Teri Walters was experiencing a hallmark process of WG following inflammation from an environmental

factor. MRE 702. The facts and data upon which Dr. Gershwin relied come from Teri Walters' medical records. MRE 702(1). (Pls' 10-25-16 MSC Br. at 4-5.) This issue was not challenged, but the peer-reviewed articles support Dr. Gershwin's opinion and methods relative to observing and analyzing high levels of ANCA in Teri Walters. MRE 702(2). Dr. Gershwin reliably applied his method of checking for high levels of ANCA because it is a hallmark of WG and Sparrow Hospital diagnosed Teri Walters with WG. MRE 702(3).

Peer reviewed articles, noted above, support Dr. Gershwin's opinion and basis regarding the role of high levels of cytoplasmic ANCA in WG onset and activity. MCL 600.2955(1)(b). The peer reviewed articles presented give no indication of any dispute in the scientific community regarding the role of ANCA in WG onset and activity, and no articles were presented that suggested a lack of general acceptance. MCL 600.2955(1)(c)(e). The authors of the cited articles are experts in the field who, outside of litigation, rely upon the abundant presence of ANCA in the onset and activity of WG. MCL 600.2955(1)(f)(g).

The COA's opinion regarding the presence of ANCA in WG onset comports with the case law interpreting admissibility of expert testimony. The COA properly considered Dr. Gershwin's method of seeking out hallmark indicators of WG onset and activity without improperly challenging Dr. Gershwin's ultimate conclusion. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95) (focus on principles and methods rather than conclusions). The COA did not search for absolute truth, require only uncontested evidence, or try to resolve a scientific dispute in analyzing the scientific discussions of high levels of ANCA in WG onset. *Id*.; *also Unger*, 278 Mich App at 217-18, 220. The COA properly accepted the undisputed presence of high levels of ANCA in Teri Walters from which Dr. Gershwin rationally derived, in part, his opinion regarding the onset of WG in Teri Walters. *Chapin*, 274 Mich App at 127. There was no conflicting expert opinion presented to refute the presence of abundant ANCA in

Teri Walters or the role of ANCA in the onset and activity of WG, but even if there had been, it would have been up to the jury to resolve such credibility issues of experts. *Unger*, 278 Mich App at 220; *Martin*, 488 Mich at 987-88. The opinion of Dr. Gershwin regarding the presence of high levels of ANCA connecting the phosphoric acid exposure to WG symptom onset is not a matter of his "own beliefs," the ANCA abundance was unchallenged, and there was presented no contradictory expert opinion or published literature regarding the role of ANCA. *Elher*, at 3, 8, 15-16. Thus, the COA's reliance on Dr. Gershwin's consideration of the presence of high levels of ANCA in Teri Walters was consistent with MRE 702, MCL 600.2955(1), and the case law interpreting those statutes.

(7) The manifestation and duration of a classic WG symptom, sinusitis. (COA Op. at 9.)

The COA appropriately relied upon the manifestation of sinusitis as support for the reliability of Dr. Gershwin's opinion temporally linking exposure to the phosphoric acid with WG symptom onset. (COA Op. at 9.) Teri Walters was diagnosed with a respiratory disease, sinusitis, that commenced only a few weeks after her exposure to phosphoric acid, and the sinusitis diagnoses were closely followed by the diagnosis of WG. (*See* Pls' 10-25-16 MSC Br. at 4-5.) Dr. Gershwin testified, with support from peer-reviewed literature, that sinusitis is a symptom of the first phase of WG rather than a cause. (*See* Pls' 10-25-16 MSC Br. at 24, 36; Dr. Gershwin Tr. at 32:3-19, **Ex. 9**; Hamidou, **Ex. 19** at 373-74 (for WG, in "the first phase, the disease is confined to the airways, causing sinusitis"); Duna, **Ex. 26** at 670 (study involving questionnaire noting sinusitis was "evaluated by a physician and subsequently attributed to WG").)

Expert testimony regarding the existence WG symptoms and the presentation of WG symptom within an appropriate immunological time frame following exposure to an inflammatory chemical assists the jury in understanding the progression of WG onset and the temporal connection between cause and effect. MRE 702. The facts and data upon which Dr.

Gershwin relied come from Teri Walters' medical records. MRE 702(1). (Pls' 10-25-16 MSC Br. at 4-5 and nn1-2.) Peer-reviewed articles support Dr. Gershwin's opinion and methods based upon tracing WG onset back to the sinusitis symptom and from there seeking an inflammatory environmental factor that Teri Walters encountered within the appropriate immunological time frame. MRE 702(2). Dr. Gershwin reliably applied his method to the facts here because the sinusitis symptom of WG onset only weeks after her exposure to the highly inflammatory phosphoric acid. MRE 702(3).

At least two peer-reviewed articles, noted above, support Dr. Gershwin's opinion and basis regarding sinusitis as an initial symptom of WG onset. MCL 600.2955(1)(b). The peer reviewed articles presented give no indication of any dispute in the scientific community regarding sinusitis as a symptom of WG onset, and no articles were presented that suggested a lack of general acceptance. MCL 600.2955(1)(c)(e). The cited articles demonstrate that experts in the field, outside of litigation, rely upon the presence of sinusitis as an indicator of the onset of WG. MCL 600.2955(1)(f)(g).

The COA's opinion regarding sinusitis as a symptom of WG comports with the case law interpreting admissibility of expert testimony. The COA properly considered Dr. Gershwin's evaluation of the time frame between WG symptoms and exposure to phosphoric acid without improperly challenging Dr. Gershwin's ultimate conclusion. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95) (focus on principles and methods rather than conclusions). The COA did not search for absolute truth, require only uncontested evidence, or try to resolve a scientific dispute regarding WG symptoms. *Id*.; *also Unger*, 278 Mich App at 217-18, 220. The COA properly relied upon undisputed evidence of the time frame between exposure and symptom onset as well as peer-reviewed literature establishing sinusitis as a symptom of WG. *Chapin*, 274 Mich App at 127 (opinion rationally derived from a sound foundation). There was

no conflicting expert opinion presented suggesting that sinusitis is not a symptom of WG, but even if there had been, it would have been up to the jury to resolve such credibility issues of experts. *Unger*, 278 Mich App at 220; *Martin*, 488 Mich at 987-88. The opinion of Dr. Gershwin regarding the immunologic response time frame between exposure and symptom onset is not a matter of his "own beliefs," it was unchallenged textbook information, and there was presented no contradictory expert opinion or published literature on the matter. *Elher*, at 3, 8, 15-16. Thus, the COA's reliance on Dr. Gershwin's consideration of the time frame between exposure and sinusitis symptom onset was consistent with MRE 702, MCL 600.2955(1), and the case law interpreting those statutes.

(8) Walters' predisposition to WG. (COA Op. at 9.)

The COA appropriately relied upon Dr. Gershwin's understanding and discussion of genetic predisposition in holding his testimony sufficiently reliable for admission. (COA Op. at 9.) The COA referenced articles addressing genetic predisposition required for WG onset. (COA Op. at 4, citing Mahr, **Ex. 18** at S-86.) Dr. Gershwin testified to the rarity of genetic predisposition to WG and that even among a large number of test subjects, none might have genetic susceptibility to the onset of WG. (Dr. Gershwin Tr. at 30:25 – 31:13, **Ex. 9**; Pls' 10-25-16 MSC Br. at 22-24.) He further testified that the phosphoric acid etching solution caused the WG onset in Walters only in combination with her genetic predisposition to WG. (Dr. Gershwin Dep. Tr. at 33-35, **Ex. 9**.) The Mahr article, written in 2006, indicates that WG had become better understood in the past 15 years as a disease arising from the "interplay among multiple genetic and environmental risk factors," and the article states that the rate of incident of WG is between 3 and 14 per million annually. (Mahr, **Ex. 18** at S-82.) The Mahr article particularly examines differing prevalence of the disease among various ethnic populations as support for the genetic susceptibility involved in WG, and the article cites considerable effort expended in studying

genetic factors of WG. (Mahr, **Ex. 18** at S-85, S-86.) Other articles similarly confirm agreement of the scientific community regarding genetic predisposition in WG onset. (Hamidou, **Ex. 19** at 373 (stating pathogenesis of WG involves genetic susceptibility and environmental factors); Lane, **Ex. 20** at 272, 274-75 (stating that genetic predisposition alone is insufficient to trigger WG absent environmental factors, and citing seven other studies of genetic factors involved in WG susceptibility).)

Expert testimony regarding the genetic susceptibility involved in WG onset assists the jury in understanding why WG does not onset among a wider population in the presence of various trigger substances, such as the phosphoric acid, and why the rarity of the disease makes it extremely difficult to develop testing models even in rodents. MRE 702. The facts and data upon which Dr. Gershwin relied come from Teri Walters' medical records regarding the diagnoses of WG. MRE 702(1). (Pls' 10-25-16 MSC Br. at 4-5 and nn1-2.) Peer-reviewed articles support Dr. Gershwin's opinion and methods of tracing WG onset back to a cause within the appropriate immunological time frame based upon the rarity of the disease resulting from the necessity of genetic predisposition. MRE 702(2). Dr. Gershwin reliably applied his method to the facts here by determining the existence of genetic predisposition through medical records and then tracing backwards from WG onset to Teri Walters' exposure to the highly inflammatory phosphoric acid weeks before. MRE 702(3).

At least three peer-reviewed articles, noted above, support Dr. Gershwin's opinion and basis regarding the genetic predisposition required for WG onset. MCL 600.2955(1)(b). The peer reviewed articles presented give no indication of any dispute in the scientific community regarding the necessity of genetic predisposition to experience WG onset, and no articles were presented that suggested a lack of general acceptance. MCL 600.2955(1)(c)(e). The cited articles demonstrate that experts in the field, outside of litigation, rely upon the role of genetic

predisposition in studying the causes and progression of WG. MCL 600.2955(1)(f)(g).

The COA's opinion regarding genetic predisposition in WG onset comports with the case law interpreting admissibility of expert testimony. The COA properly considered Dr. Gershwin's evaluation of the existence and import of genetic predisposition in WG onset and progression, along with Teri Walters' diagnosis of WG, without improperly challenging Dr. Gershwin's ultimate conclusion. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95) (focus on principles and methods rather than conclusions). The COA did not search for absolute truth, require only uncontested evidence, or try to resolve a scientific dispute regarding WG genetic predisposition. *Id.*; *also Unger*, 278 Mich App at 217-18, 220. The COA properly relied upon undisputed evidence of WG diagnosis as to Teri Walters as well as peer-reviewed literature establishing the role of genetic predisposition in WG onset. *Chapin*, 274 Mich App at 127 (opinion rationally derived from a sound foundation). There was no conflicting expert opinion presented to refute genetic susceptibility in WG, but even if there had been, it would have been up to the jury to resolve such credibility issues of experts. *Unger*, 278 Mich App at 220; *Martin*, 488 Mich at 987-88. The opinion of Dr. Gershwin regarding the need for genetic predisposition for WG onset and the fact of genetic predisposition in Teri Walters are not matters of his "own beliefs," the diagnosis and predisposition were unchallenged, and there was presented no contradictory expert opinion or published literature on the matter. *Elher*, at 3, 8, 15-16. Thus, the COA's reliance on Dr. Gershwin's consideration of the effect of genetic predisposition in evaluating whether phosphoric acid was a potential WG triggering chemical was consistent with MRE 702, MCL 600.2955(1), and the case law interpreting those statutes.

(9) The support of scientific and medical literature either directly or by analogy. (COA Op. at 9.)

The COA properly accepted Dr. Gershwin's reliance upon scientific literature, either directly

or by analogy, in holding his testimony regarding phosphoric acid as the trigger for WG onset in Teri Walters to be sufficiently reliable for admission. (COA Op. at 9.) The COA addressed the issue of whether phosphoric acid had been specifically connected in medical literature to the onset of WG. (COA Op. at 11.) The COA referenced articles demonstrating statistically significant associations between exposure to pesticides and the onset of WG, and the COA referenced articles explaining that phosphorous is a key component of pesticides. (COA Op. at 4-5, 11, citing Lane, **Ex. 25** at 814-15 (demonstrating association between pesticides and WG); Duna, **Ex. 26** at 669 (statistically significant association between WG and exposure to fumes, particulate materials, particulate materials from construction, and occupational exposure to pesticides); *Organophosphorus Insecticides*, **Ex. 29** at 1 (organophosphorus insecticides have accounted for a large share of all United States insecticides); *Organophosphate Pesticides,* **Ex. 27** at 1 (organophosphate pesticides account for about half of insecticides used in the United States); *Organo-Phosphorus Pesticides*, **Ex. 27** at 2005 (addressing prevalence of pesticides containing phosphorous); *see also Organophosphorus Insecticides*, **Ex. 36** at 1 (identifying crisis responses to exposure to phosphoric insecticides such as difficulty breathing, paralysis, and seizures); Petty, **Ex. 37** at 62 (discussing use of organic phosphate insecticides and their toxicity); Blanc-Lapierre, **Ex. 38** at 1086 (discussing widespread use of organophosphates in pesticides since 1970s); Wang, **Ex. 39** at 588, 595 (discussing widespread use of organophosphorus pesticides and organ damage effected in rats).)

Plaintiffs have also provided this Court with a substantial analysis of scientific literature demonstrating a wide variety of substances linked to the onset of WG, that phosphorous shares characteristics with substances linked to WG onset, phosphorous is an important component of various substances linked to WG onset, phosphorous is capable of extreme biological trauma and inflammation that is the initial step in the onset of WG, abnormal levels of phosphorous have

been found in the body of a deceased WG patient, and phosphorous plays a critical role in the body's immunological response leading to the onset of WG. (Pls' 10-25-16 MSC Br. at 27-36.) One article specifically addresses "acid phosphatase," which has been associated with the initiation and relapse of WG, and notes that antibodies to the acid phosphatase are present in WG patients. (Chen, **Ex. 23** at A293, A295.)

Scientific articles support a connection between WG onset and a broad variety of substances, including farming products and occupational solvent exposure (Lane, **Ex. 25** at 814); toxic substances and silica (Hamidou, **Ex. 19** at 373); silica, organic solvents, and pesticides (Mahr, **Ex. 18** at S-82, S-86 to S-88); statistically significant associations with fumes, particulate materials, construction particulates, and pesticides (Duna, **Ex. 26** at 669, 673); hydrocarbons and farming (Lane, **Ex. 20** at 272). (Pls' 10-25-16 MSC Br. at 28-29.)

Dr. Gershwin analogized substances such as silica to phosphoric acid based upon shared characteristics of isoelectric focusing and electrophilic properties, which are important properties in the onset of autoimmune disease. (Pls' 10-25-16 MSC Br. at 29-31, citing articles including Gershwin, **Ex. 30** at 209-16 (studying impact of electrophilic agents in initiating autoimmune disease); Husain, **Ex. 31** at 14-15 (electrophilic and reactive phosphoric compounds used in nerve agent weapons); Metcalf, **Ex. 32** at 340 (describing electrophilic nature of phosphorous and its reactivity and usefulness in insecticides); Yamashita, **Ex. 33** at 1017, 1019, 1024 (showing competing characteristics of silica and phosphorous); Smith, **Ex. 34** at 1155 (other compounds take on electrophilic properties in the presence of silica); OSHA, **Ex. 35** at 234 (silica induces the activity of phosphorous molecules in autoimmune processes).)

The COA analyzed the *Chapin* opinion in which there were studies actually *denying* any correlation between asbestos in brake products and mesothelioma, but the expert in that case was permitted to testify. (COA Op. at 12.) Unlike *Chapin*, Plaintiffs produce numerous articles

addressing chemicals containing the same underlying substances or substances similar in characteristics to the phosphoric acid to which Teri Walters was exposed. Such articles refer to phosphoric substances, such as pesticides, as well as fumes and solvents, which categories generally include a substance such as phosphoric acid, and electrophilic substances such as silica, which share characteristics with phosphorus. Also unlike *Chapin*, Defendants have produced no articles refuting a connection between phosphoric acid and the onset of WG.

The *Walters* case is far easier to resolve than the *Chapin* case. In *Chapin*, the parties agreed that asbestos causes mesothelioma in humans, but they disagreed upon the sufficiency of exposure. *Chapin*, 274 Mich App at 130-31. The **unique factor** in *Chapin* was the sufficiency of exposure to a particular substance, asbestos, and the **common factor** in *Chapin* is that humans develop mesothelioma from such exposure. In *Walters*, the **unique factor** is *genetic predisposition*, and the **common factor** is that a wide range of chemicals and substances have been linked to the airway inflammation necessary to onset WG. The **unique factor** in *Walters* is met because the medical records establish that Teri Walters is one of those 3 in a million people susceptible to WG. Defendants have treated the chemical trigger in *Walters* as if it were a unique factor as in *Chapin*, where *only* asbestos could generate the disease. Here, Dr. Gershwin and the Plaintiffs have presented the Court with dozens of scientific articles demonstrating that ***many substances and types of substances are capable of instigating the onset of WG***. What Dr. Gershwin has demonstrated, through his testimony and documents, is that compared to the many chemicals that have been associated with WG onset, ***phosphoric acid is similar in characteristics but much more potent***, and compared to the types of exposure other WG patients have experienced, ***not one article comes close to describing the lengthy, intense, and virtually clinical exposure that Teri Walters experienced by sleeping with the offending substance in her mouth overnight***. *Walters* is an easier case for admissibility than *Chapin* because the unique

factor, genetic predisposition is met, and the common factor, sufficient exposure to an inflammatory substance, is easily met by the incredibly intense exposure to phosphoric acid that is potent enough, as Defendants admitted in testimony, to destroy the hardest substance in the body: teeth. Defendants have cited no evidence of Teri Walters' exposure to any other substance capable of initiating WG. Instead, Defendants' scrutiny of the scientific literature to find a study specifically dealing with phosphoric acid is like sorting through articles stating that firearms produce lethal projectiles in search of an article discussing whether a shoulder-mounted rocket launcher dispenses a lethal projectile. It is just bigger. It is just more powerful. Of course it can do the job. Phosphoric acid eats through the hardest substance in the body, and Dr. Gershwin's essential point is that it can cause the necessary WG inflammation that is caused by pesticides, *which we put on our food*, or a variety of other triggers that are also less potent.

Expert testimony regarding the capacity of phosphoric acid to initiate WG onset assists the jury in understanding the connection between Defendants' negligent dispensing of phosphoric acid and Teri Walters onset of WG after sleeping with the acid in her mouth. MRE 702. The facts and data upon which Dr. Gershwin relied come from Teri Walters' testimony and affidavit regarding her use of the phosphoric acid on February 11, 2011, followed by medical records regarding the diagnoses of WG, and from the Ultradent Safety Data Sheet. MRE 702(1). (Pls' 10-25-16 MSC Br. at 3-5 and nn1-2; Safety Data Sheet at 1-2, **Ex. 22**.) Peer-reviewed articles support Dr. Gershwin's opinion and methods of investigating backward from the onset of WG to locate substances capable of initiating WG and in analogizing phosphoric acid to other substances with similar characteristics or components that have been associated with the onset of WG. MRE 702(2). Dr. Gershwin reliably applied his method to the facts here by determining the time frame of WG onset through medical records and then tracing backwards from WG onset to Teri Walters' exposure to the highly inflammatory phosphoric acid weeks before. MRE 702(3).

Numerous peer-reviewed articles, noted above, support Dr. Gershwin's opinion and basis regarding the capacity of a substance like phosphoric acid to provide the airway inflammation required for WG onset. MCL 600.2955(1)(b). The peer reviewed articles presented give no indication of any dispute in the scientific community regarding the capacity of phosphoric acid to cause WG onset, the articles establish a general acceptance of a wide variety of substances capable of the necessary inflammatory insult to trigger WG, and no articles were presented that refute the capacity of intense exposure to phosphoric acid to onset WG. MCL 600.2955(1)(c)(e). The cited articles demonstrate that experts in the field, outside of litigation, rely upon studies of general types of WG triggering substances, such as "fumes," "particulates," "pesticides," "solvents," "farming products," and the like, rather than focusing on whether a particular chemical can cause the necessary inflammation, and experts in the field conduct research retrospectively to consider what types of chemicals WG patients have encountered. MCL 600.2955(1)(f)(g).

The COA's opinion regarding use directly or by analogy of scientific literature regarding the triggering agent in WG onset comports with the case law interpreting admissibility of expert testimony. The COA properly considered Dr. Gershwin's evaluation of the characteristics and potency of phosphoric acid relative to other types of substances linked to the onset of WG in scientific articles without improperly challenging Dr. Gershwin's ultimate conclusion. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95) (focus on principles and methods rather than conclusions). The COA did not search for absolute truth, require only uncontested evidence, or try to resolve a scientific dispute regarding the scope of WG triggering agents. *Id*.; *also Unger*, 278 Mich App at 217-18, 220. The COA properly relied upon undisputed evidence of WG diagnosis as to Teri Walters, information regarding the characteristics and potency of phosphoric acid, and peer-reviewed literature describing the characteristics of substances capable

of causing WG onset. *Chapin*, 274 Mich App at 127 (opinion rationally derived from a sound foundation). There was no conflicting expert opinion presented to refute the types of chemicals capable of initiating WG, but even given the affidavit of Dr. Mohan stating to "a reasonable degree of medical certainty" that the etching solution did not cause the WG onset and that Dr. Mohan is not aware of literature regarding etching solution, it is up to the jury to resolve such credibility issues of experts. *Unger*, 278 Mich App at 220; *Martin*, 488 Mich at 987-88. The opinion of Dr. Gershwin regarding the triggering agent for WG onset is not a matter of his "own beliefs," the peer-reviewed literature supports triggering agents that are similar but less potent, and there was presented no expert opinion providing a contradictory cause of WG in Teri Walters or providing any contradictory published literature on the matter. *Elher*, at 3, 8, 15-16. Thus, the COA's reliance on Dr. Gershwin's use of scientific literature directly or by analogy regarding the triggering agent in WG onset was consistent with MRE 702, MCL 600.2955(1), and the case law interpreting those statutes.

The COA correctly determined that this case is highly distinguishable from *Elher*, in which the expert based his opinion solely on his own personal beliefs. (COA Op. at 15.) Here, the reliability of Dr. Gershwin's principles and methodologies is supported by numerous factors such that his testimony was admissible and any challenge to the weight of his opinion is a matter for jury resolution. (COA Op. at 15.) The COA recognized a "logical sequence of cause and effect" and analyzed at least nine different factors establishing that a jury may conclude that "more likely than not" the phosphoric acid was "substantial factor" in causing the onset of WG in Teri Walters. *Skinner*, 445 Mich at 164-65 and n8.

II. The Trial Court Erred In Its Application Of MCL 600.2955(1) And MRE 702 And Abused Its Discretion In Granting The Defendants' Motion To Preclude The Plaintiffs' Expert Testimony.

The Circuit erred in its application of MCL 600.2955(1) and MRE 702 and abused its

discretion in granting Defendants' motion to preclude the testimony of Dr. Gershwin. Defendants filed a motion in limine from on September 3, 2013, only four weeks before a scheduled trial. The Circuit worked through the afternoon of September 18, 2013 and into the night and again early in the morning of September 19, 2013 to review the parties' briefs and the articles before a hearing on September 19, 2013. (Hrg. Tr. at 3-4, **Ex. 11**.) Despite these efforts, the Circuit erred by requiring uncontested scientific opinions and by establishing an overly rigorous admissibility standard demanding definitive proof of causation prior to trial, which is not contemplated under MRE 702, MCL 600.2955(1), or the relevant interpretive case law, which the Circuit said it was following. (Hrg. Tr. at 19, **Ex. 11**.)

The Circuit erred and abused its discretion by ignoring scientific literature, factual support, and other considerations analyzed by the COA as well as by ignoring or misconstruing the following key components of Dr. Gershwin's opinions and methods. (i) Scientific articles establish connections between WG onset and environmental factors such as phosphorus, phosphates, fumes, solvents, and other chemicals sharing characteristics with phosphoric acid. (ii) Genetic predisposition is necessary for the onset of WG. (iii) Teri Walters' WG onset occurred subsequent to her exposure to phosphoric acid, and Dr. Gershwin's testified her WG was a result of that exposure. (iv) The pathogenesis of WG is well documented in the scientific literature. (v) The scientific literature supports Dr. Gershwin's analysis of the role of cytoplasmic ANCA in WG onset and development. (vi) Causation of WG onset is supported in the scientific literature even though experimental testing is not feasible. Plaintiffs rely on the previous section in support of various articles, factual data, and other considerations overlooked by the Circuit in precluding Dr. Gershwin's testimony. Six key errors of the Circuit are examined below.

1. Environmental Factors

The Circuit contended that Dr. Gershwin's use of the phrase "environmental factors" was too

broad. (Hrg. Tr. at 21, 29, **Ex. 11**.) However, Dr. Gershwin testified that there are various "well-defined environmental factors in autoimmunity," which he proceeded to list as including "materials that alter the mucosal airway," "superantigens," silica exposure, chemicals, hydrocarbons, solvents, and pesticides. (Dr. Gershwin Tr. at 8:10 – 9:2, **Ex. 9**.) The Circuit disregarded the value of studies of other chemicals by stating that it could not find reference in the literature to phosphorous, phosphate, phosphoric acid, fertilizers, solvents, chemicals, pesticides, or acids that have been linked to the onset of WG. (Hrg. Tr. at 26-27, 29, **Ex. 11**.) In fact, as to articles addressing phosphate and phosphorus, the Circuit said "there were no articles" and "I was a little surprised that there weren't any." (Hrg. Tr. at 26, **Ex. 11**.) Scientific articles submitted by Plaintiffs support a connection between WG onset and a broad variety of substances, including those listed by Dr. Gershwin: farming products and occupational solvent exposure (Lane, **Ex. 25** at 814); toxic substances and silica (Hamidou, **Ex. 19** at 373); silica, organic solvents, and pesticides (Mahr, **Ex. 18** at S-82, S-86 to S-88); statistically significant associations with fumes, particulate materials, construction particulates, and pesticides (Duna, **Ex. 26** at 669, 673); hydrocarbons and farming (Lane, **Ex. 20** at 272). (Pls' 10-25-16 MSC Br. at 28-29.) Another article specifically addresses "acid phosphatase," which has been associated with the initiation and relapse of WG, and notes that antibodies to the acid phosphatase are present in WG patients. (Chen, **Ex. 23** at A293, A295.)

Furthermore, the Circuit had before it articles demonstrating statistically significant associations between exposure to pesticides and the onset of WG and articles explaining that phosphorous is a key component of pesticides. (COA Op. at 4-5, 11; Lane, **Ex. 25** at 814-15 (demonstrating association between pesticides and WG); Duna, **Ex. 26** at 669 (statistically significant association between WG and exposure to fumes, particulate materials, particulate materials from construction, and occupational exposure to pesticides); Metcalf, **Ex. 32** at 340

(describing electrophilic nature of phosphorous and its reactivity and usefulness in insecticides); *Organophosphorus Insecticides*, **Ex. 29** at 1 (organophosphorus insecticides have accounted for a large share of all United States insecticides); *Organophosphate Pesticides*, **Ex. 27** at 1 (organophosphate pesticides account for about half of insecticides used in the United States); *Organo-Phosphorus Pesticides*, **Ex. 27** at 2005 (addressing prevalence of pesticides containing phosphorous); *Organophosphorus Insecticides*, **Ex. 36** at 1 (identifying crisis responses to exposure to phosphoric insecticides such as difficulty breathing, paralysis, and seizures); Petty, **Ex. 37** at 62 (discussing use of organic phosphate insecticides and their toxicity); Blanc-Lapierre, **Ex. 38** at 1086 (discussing widespread use of organophosphates in pesticides since 1970s); Wang, **Ex. 39** at 588, 595 (discussing widespread use of organophosphorus pesticides and organ damage effected in rats).)

The Circuit debated the relevance of silica, which is next to phosphorus on the periodic chart of elements, to Walters' exposure to phosphoric acid. (Hrg. Tr. at 20, 29, **Ex. 11**.) However, silica shares key characteristics with phosphoric acid relevant to the inflammation and process of WG onset and supported by scientific literature as discussed previously. (*See* § I(9), *supra*.)

The Circuit committed error and abused its discretion by overlooking every article presented by Plaintiffs that established connections between WG onset and phosphorus, phosphates, fumes, solvents, and other chemicals sharing characteristics with phosphoric acid.

2. Genetic Predisposition

The Circuit also committed error and abused its discretion in ignoring the key feature of genetic susceptibility in WG onset. Plaintiffs have already examined the facts and articles supporting Dr. Gershwin's reliance on genetic predisposition in analyzing the cause of WG onset in Teri Walters. (*See* § I(8), *supra*.) The Circuit missed the importance of genetic predisposition and its impact on the scientific community's common method of retrospectively analyzing

triggers for WG onset in patients rather than attempting to affirmatively test for such triggers. (Hrg. Tr. at 23, 25, 28, **Ex. 11**.)

Dr. Gershwin testified that 100 people could be exposed to etching solution and not develop WG. The genetic predisposition to WG is so rare that it is likely no one in the test population would develop WG even with an appropriate triggering agent. (Dr. Gershwin Tr. at 30:25 – 31:13, **Ex. 9**.) The Circuit expressed concern that people could be exposed to a highly potent electrophilic solution but those people might not develop WG. (Hrg. Tr. at 23, 25, **Ex. 11**; Dr. Gershwin Tr. at 30 - 31, **Ex. 9**.) That concern stemmed from the Circuit's failure to consider the well-supported conclusion that WG requires a rare genetic predisposition. Instead, the court erroneously assumed that a caustic, electrophilic solution is not a reliable trigger for Walters' WG onset because the chemical might not trigger WG in many subjects.

Failure to properly analyze genetic predisposition in the onset of WG caused the court to erroneously draw a distinction between the reasoning applied in *Chapin*, 274 Mich App 122, and to reject the reliability of Dr. Gershwin's opinions on WG. The Circuit noted that in *Chapin*, the court determined that asbestos affects all people the same way, and the Circuit contrasted that with its misunderstanding of Dr. Gershwin's testimony that any number of people could be exposed to electrophilic solutions without developing WG. (Hrg. Tr. at 22-23, **Ex. 11**.) Asbestos-related diseases are not premised upon *genetic predisposition*, while WG only afflicts those with a genetic predisposition. Consequently, the Circuit minimized the reliability of Dr. Gershwin's opinion by improperly comparing a non-genetic disease to a genetic disease. The Circuit also ignored the variety of substances associated with WG onset and attempted to fit *Walters* into the *Chapin* factual circumstance in which only one substance could result in the subject disease. As discussed in a preceding section of this brief, the *Walters* case is far easier to resolve than the *Chapin* case, but the Circuit made it more difficult by ignoring differences between the two

diseases. (*See* § I(9), *supra*.)

The Circuit committed error and abused its discretion by overlooking a key feature of WG onset, genetic predisposition, which caused the Circuit to misapply *Chapin*.

3. Cause Precedes Effect

The Circuit denied that Dr. Gershwin's analysis satisfied the temporality factor of the SBH test and that Teri Walters' exposure to phosphoric acid (cause) preceded the onset of WG (effect). (Hrg. Tr. at 24-25, **Ex. 11**.) The Circuit read Dr. Gershwin's testimony to state that Walters might have experienced WG onset regardless of the phosphoric acid. Significantly, it also remarked that it might be misunderstanding Dr. Gershwin's testimony. (Hrg. Tr. at 25, **Ex. 11**.) The Circuit committed an abuse of discretion in misinterpreting Dr. Gershwin's unwavering testimony that Walters's exposure to phosphoric acid etching solution triggered the onset of WG.

Defense counsel asked Dr. Gershwin at his deposition if it was Dr. Gershwin's opinion that "without the etching solution Mrs. Walters would not have developed Wegener's." (Dr. Gershwin Tr. at 34:15-17, **Ex. 9**.) This question was hypothetical in that it asked Dr. Gershwin to testify what would happen if Walters *was not* exposed to etching solution even though the facts as they exist establish that Walters *was* exposed to the solution. Dr. Gershwin gave the following response to defense counsel's subtle hypothetical question:

> Well, she wouldn't have developed Wegener's here at this time. I mean, I can say that she would not have got Wegener's had it not been for the etching solution. I can't say she wouldn't have gotten Wegener's in the future. It's possible. But she certainly wouldn't have got Wegener's at this moment. (Dr. Gershwin Tr. at 34:18-23, **Ex. 9**.)

The Circuit misinterpreted this testimony as meaning that Walters may have presently developed WG regardless of the etching solution. Dr. Gershwin reaffirmed that the reason for the onset of WG in this case is that Walters was exposed to phosphoric acid etching solution. (Dr. Gershwin Aff., dated Oct. 17, 2013, ¶ 1, **Ex. 43**.) Dr. Gershwin clarified to the Circuit that the

remainder of his testimony was in response to the hypothetical nature of defense counsel's question about what might have happened to Walters *without* being exposed to etching solution. (Dr. Gershwin Aff., dated Oct. 17, 2013, ¶ 1, **Ex. 43**.) Dr. Gershwin's testimony was and is that some other environmental factor in the future might have triggered WG if Walters had hypothetically never developed WG from encountering the etching solution. (Dr. Gershwin Aff., dated Oct. 17, 2013, ¶ 1, **Ex. 43**.) Such a hypothetical scenario does not undermine Dr. Gershwin's testimony that Walters was actually exposed to etching solution and that exposure caused the onset of WG. (Dr. Gershwin Aff., dated Oct. 17, 2013, ¶ 1, **Ex. 43**.)

The Circuit committed error and abused its discretion by misinterpreting the well-supported fact that Teri Walters' WG onset occurred subsequent to her exposure to phosphoric acid and Dr. Gershwin's unequivocal opinion was that she developed WG as a result of that exposure.

4. Pathogenesis of WG

The Circuit attacked Dr. Gershwin's reliability based upon the Circuit's belief that his testimony regarding the scientific community's general acceptance of the pathogenesis of WG was incorrect. (Hrg. Tr. at 29-30, **Ex. 11**.) Dr. Gershwin testified very clearly as to the pathological process of WG onset and development from inflammation of the airways to the release of cytoplasmic antigens from neutrophil to release of antibodies causing the neutrophils to slow and adhere to the blood vessels creating additional inflammation that compounds and clogs the vessels. (Dr. Gershwin Tr. at 12:22-24, 13:7-12, 14:14-15:10, 15:16-16:13, **Ex. 9**.) Two scientific articles in particular establish the scientific community's accepted process of the onset and progression of WG that is completely consistent with Dr. Gershwin's testimony. (Chen, **Ex. 23** at A293, A296 and Fig. 1 ("ANCA are serological hallmarks" for small vessel vasculitis such as WG; environmental factors stimulate inflammation, generating expression of neutrophil adhesion molecules and movement of ANCA antigens to the cell surfaces, resulting in the

binding of ANCA with ANCA antigens on the cell surfaces, leading to the adherence of the neutrophils to blood vessel walls); Sibelius, **Ex. 24** at 497, 502 (describing the pathology of WG onset starting with respiratory "burst" or inflammation).)

The Circuit committed error and abused its discretion by considering Dr. Gershwin unreliable based upon the Circuit's misunderstanding of the testimony and supporting articles regarding the pathogenesis of WG.

5. Cytoplasmic ANCA

The Circuit next erred in challenging whether Dr. Gershwin had properly differentiated between different types of ANCA when testifying why the ANCA, an antibody, is made. The Court did note uncertainty about its analysis. (Hrg. Tr. at 30, **Ex. 11**.)

ANCAs are "anti-neutrophil cytoplasmic antibodies." (Sibelius, **Ex. 24** at 497.) Cytoplasmic ANCA (c-ANCA) is a seromarker of WG that is 95 percent specific to WG. *Id*. Proteinase 3 (PR3) is the target of the c-ANCA, meaning it is the substance that the antibody c-ANCA attacks. *Id*. In response to inflammation, neutrophils manifest PR3, which is then targeted by c-ANCA. *Id*. The importance of the presence of c-ANCA is that it indicates the onset of WG and is involved in the progression of the disease. *Id*. at 497, 502.

The Sibelius article is consistent with Dr. Gershwin's testimony on the pathology of WG and the presence of a "cytoplasmic antigen," such as PR3, and that the antibody generated was ANCA. (Dr. Gershwin Tr. at 15:19 – 16:10, **Ex. 9**.) Dr. Gerswhin characterized the antigens as "cytoplasmic antigens," which necessarily means that the antibody responsive to such an antigen is a cytoplasmic antibody, or c-ANCA. Consequently, Dr. Gershwin described the pathology of WG arising from an ANCA response to a cytoplasmic antigen consistent with the Sibelius article.

The Circuit committed error and abused its discretion by considering Dr. Gershwin

unreliable based upon the Circuit's misunderstanding of the testimony and supporting articles regarding cytoplasmic ANCA.

6. Causation is not "science fiction" even though experimental testing is not feasible.

The Circuit mischaracterized Dr. Gershwin's testimony by holding that Dr. Gershwin agreed that causality in the WG arena "sounds like science fiction" and that Dr. Gershwin "was essentially saying we're not there yet in medicine." (Hrg. Tr. at 25-26, **Ex. 11**.) Defense counsel had earlier asked Dr. Gershwin for a peer-reviewed article specifically showing that phosphoric acid has caused WG, and Dr. Gershwin responded that many epidemiological studies exist showing a *variety of chemicals*, including those containing phosphorus, are associated with WG onset. (Dr. Gershwin Tr. at 24:16 – 25:11, **Ex. 9**.) Defense counsel then returned to the issue of whether case studies had been done specifically involving etching solutions or phosphoric acid and WG onset, and Dr. Gershwin repeated that studies on people are unethical. (Dr. Gershwin Tr. at 28:16-25, **Ex. 9**.) Defense counsel again requested a study showing phosphoric acid causing WG onset, and Dr. Gershwin explained that there is a scientifically acceptable mechanism of action at work but a study giving people etching solution would fail because the genetic predisposition component of WG would make it impossible to know whether the test subjects, including animal subjects, were susceptible even with a valid WG trigger chemical. (Dr. Gershwin Tr. at 30:13 – 31:13, **Ex. 9**.) Thus, Dr. Gershwin responded to a volley of questions repeatedly aimed at testing phosphoric acid on human or animal subjects before the "science fiction" response that attracted the Circuit's attention.

In the "science fiction" passage to which the Circuit refers, defense counsel asked Dr. Gershwin how he knows that phosphoric acid caused the onset of WG in Teri Walters. Dr. Gershwin responded to this and a follow-up question, but he also explained the difficulty of inducing in animals for testing purposes diseases much more common than WG and that he

anticipates a time when the DNA of a WG patient like Teri Walters could be transferred to a mouse and that mouse could be studied, even though right now one "might say that's science fiction" and it would cost billions of dollars. (Dr. Gershwin Tr. at 34:18 – 36:25, **Ex. 9**.) The Circuit overlooked Dr. Gershwin's repeated attempts to explain to defense counsel the fallacy of looking for articles in which scientists intentionally exposed patients to harmful chemicals to see if WG would onset. The Circuit also overlooked Dr. Gershwin's explanation of the current difficulties in transferring genes to animals for testing as a reason that scientists examine WG onset by comparison to a *wide variety* of chemicals that are already associated with WG onset. The Circuit focused on Dr. Gershwin's testimony regarding the seemingly "science fiction" nature of transferring genes for animal testing, but *the Circuit mischaracterized this as an admission that the scientific community has no ability to examine the causes of WG onset*. (Hrg. Tr. at 25-26, **Ex. 11**.)

The Circuit missed the entire point: ethics and genetic susceptibility prevent effective experimental testing, but scientists have identified a broad array of chemicals associated with WG onset, and phosphoric acid is similar to, *but more potent than*, many such chemicals. Dr. Gershwin testified that it is unethical to perform experimental testing and expose patients to etching solution to evaluate the onset of WG, and the absence of experimentation necessitates the use of analogy to peer-reviewed retrospective studies of naturally occurring incidences of WG arising from exposure to a variety of substances. (Dr. Gershwin Tr. at 21:23 - 22:2, 23:1-13, 23:24 – 24:3, 28:20-23, **Ex. 9**.) The court in *Chapin* recognized the ethical problems with experimental tests involving dangerous substances, such as asbestos. *Chapin*, 274 Mich App at 134. (*See also* discussion of retrospective analysis in scientific literature, *Robelin v Spectrum Health Hosps*, unpub'd op *per curiam* of the Court of Appeals, issued Sept 10, 2009 (Dkt. No. 279780); 2009 Mich App LEXIS 1865 (**Ex. 46**); *Robelin v Spectrum Health Hosps*, 488 Mich

1000 (2010) and *Clerc v Chippewa County War Mem Hosp*, unpub'd op *per curiam* of the Court of Appeals, issued Nov 14, 2013 (Dkt. No. 307915); 2013 Mich App LEXIS 1823 (**Ex. 47**), where prospective testing in impossible or unethical, *see* Pls' 10-25-16 MSC Br. at 20-22.).)

Dr. Gershwin further testified that with diseases much more common than WG, it can take decades to find animal models to test the causes of the disease. (Dr. Gershwin Tr. at 35:16 – 36:2, **Ex. 9**.) Moreover, replicating multiple genes affecting genetic susceptibility in WG is vastly more complex than transfer of only a *single gene* in multi-billion-dollar cancer research. (Dr. Gershwin Tr. at 35:16 – 36:25, **Ex. 9**.)[3] Scientific articles verify Dr. Gershwin's testimony by confirming in January 2012, "[t]o date there are no good models that replicate the granulomatous lesions found in . . . Wegener's." (Salama, **Ex. 44** at 1; *see also* Kallenberg, **Ex. 45** at 399 ("[u]nfortunately, an animal model for PR3-ANCA—associated Wegener's granulomatosis is not yet available").) Dr. Gershwin's testimony was accurate and reliable regarding the unavailability of experimental testing and the scientific community's practice of identifying a wide variety of WG triggers through retrospective analysis.

The Circuit committed error and abused its discretion by mischaracterizing Dr. Gershwin's testimony regarding the seemingly "science fiction" nature of anticipated animal testing and by ignoring Dr. Gershwin's consistent testimony regarding the current methods for examining WG causation through similar substances already associated with WG onset.

---

[3] This testimony is relevant to an unpublished Louisiana federal case, *Frischhertz v SmithKline Beacham Corp* (ED La 2012), filed with Defendants' November 15, 2016 reply brief in support of leave to this Court. (Defs' Leave Reply at 2-3.) *Frischertz* dealt with expert testimony on whether a particular substance could cause a *genetic alteration* resulting in hand abnormality. *Walters* does not involve *genetic alteration* but the triggering of a *genetic predisposition* through inflammation. Dr. Gershwin's testimony regarding the complexities transferring or modifying genes demonstrates the great separation between *genetic alteration* in *Frischertz* and triggering an existing *genetic predisposition* in *Walters*. Moreover, one expert in *Frischertz* changed his mind about whether EF had a heart defect at all, there were no articles linking chemicals to the proposed medical event, another expert was not qualified, and the same expert "lumped" the congenital malformations together in a manner considered unreliable in the scientific community. None of these drawbacks apply to Dr. Gershwin's testimony in *Walters*.

The Circuit failed to take into account the substantial support for the reliability of Dr. Gershwin's methods and opinions as is well established in the COA opinion and the discussion in section I of this brief. Moreover, the Circuit committed error and abused its discretion with respect to at least six key areas affecting the core of Dr. Gershwin's methods, opinions, and reliability. The Circuit erred and abused its discretion by overlooking every article presented by Plaintiffs that established connections between WG onset and phosphorus, phosphates, fumes, solvents, and other chemicals sharing characteristics with phosphoric acid. The Circuit erred and abused its discretion by overlooking a key feature of WG onset, genetic predisposition. The Circuit erred and abused its discretion by misinterpreting the well-supported fact that Teri Walters' WG onset occurred subsequent to her exposure to phosphoric acid and Dr. Gershwin's unequivocal opinion was that she developed WG as a result of that exposure. The Circuit erred and abused its discretion by considering Dr. Gershwin unreliable based upon the Circuit's misunderstanding of the testimony and supporting articles regarding the pathogenesis of WG and regarding cytoplasmic ANCA. The Circuit erred and abused its discretion by mischaracterizing Dr. Gershwin's testimony regarding the seemingly "science fiction" nature of anticipated animal testing and by ignoring Dr. Gershwin's consistent testimony regarding the current methods for examining WG causation through similar substances already associated with WG onset.

The Circuit decision is not within the "range of principled outcomes" because it disregards all the evidence presented on key issues, ignored the scientific articles, misconstrues the facts, and mischaracterizes the expert's testimony, reasoning, and opinion. *Barnett,* 478 Mich at 158. Moreover, it was legal error to preclude admissibility of expert testimony where the Circuit failed to adequately apply MRE 702, MCL 600.2955(1), and case law such as *Chapin* to the evidence before it because the Circuit overlooked the evidence, mischaracterized the evidence, misinterpreted the meaning of the evidence, or failed to consider the scientific literature. *Gilbert,*

470 Mich at 782 (mandating a searching inquiry of underlying data and expert interpretation); *Lukity*, 460 Mich at 488 ("whether a rule of evidence or statute precludes admissibility of the evidence" is a preliminary question of law subject to *de novo* review). The Circuit improperly called upon Plaintiffs to negate all other possible causes, *even where none were proposed*, and improperly demanded absolute certainty as to the phosphoric acid's role in causation. *Skinner*, 445 Mich at 166. The Circuit improperly focused on Dr. Gershwin's ultimate conclusions rather than the soundness of his reasoning and his reliance on methods and information within the scientific literature. *Chapin*, 274 Mich App at 127 (quoting *Daubert*, 509 US at 594-95); *Unger*, 278 Mich App at 217-18, 220. The Circuit abandoned its "gatekeeper" role and improperly insisted on absolute truth while attempting to resolve a scientific dispute between the parties about whether phosphoric acid is capable of triggering WG onset even though Defendants produced no evidence to the contrary. *Chapin*, 274 Mich App at 127. Then, the Circuit improperly removed from the jury a matter of credibility and weight of evidence. *Martin*, 488 Mich at 987-88.

CONCLUSION

For the reasons set forth above, Plaintiffs respectfully request that this Honorable Court deny Defendants' Application for Leave to Appeal (after Remand) or affirm the COA opinion.

Respectfully submitted,

HERTZ SCHRAM PC

Dated: June 21, 2017

By: /s/ Daniel W. Rucker
Daniel W. Rucker (P67832)
Attorney for Plaintiffs/Appellees

INDEX OF EXHIBITS

Exhibit 20: Lane, S., Watts, R., and Scott, D., *Epidemiology of Systemic Vasculitis* (Curr. Rheumatology Reports, Vo. 7, 2005)

Exhibit 21: Lee, Y.H., *et al*., *The protein tyrosine phosphatase nonreceptor 22 C1858T polymorphism and vasculitis: a meta-analysis* (Mol Biol Rep, ed. 39, 2012)

Exhibit 22: Safety Data Sheet

Exhibit 23: Chen, M. and Kallengberg, C., *The environment, geoepidemiology and ANCA-associated vasculitides* (Autoimmunity Reviews ed. 9, 2010)

Exhibit 24: Sibelius, U., *et al*., *Wegener's Granulomatosis: Anti-proteinase 3 Antibodies Are Potent Inductors of Human Endothelial Cell Signaling and Leakage Response* (J. Exp. Med., Vol. 187, No. 4, Feb. 16, 1998)

Exhibit 25: Lane, S., Watts, R., *et. al*, *Are Environmental Factors Important in Primary Systemic Vasculitis?* (Arthritis & Rheumatism, Vol. 48. No. 3, 2003)

Exhibit 26: Duna, G.F., Cotch, M.F., *et. al*, *Wegener's granulomatosis: Role of environmental exposures* (Clinical and Experimental Rheumatology, Vol. 16, 1998)

Exhibit 27: Betteridge, D., Thompson, M., Baker, A.D., and Kemp, N.R., *Photoelectron Spectra of Phosphorus Halides, Alkyl Phosphites and Phosphates, Organo-Phosphorus Pesticides, and Related Compounds* (Analytical Chemistry, Vol. 44 No. 12, 1972)

Exhibit 28: GoodGuide, *Organophosphate Pesticides: Dialkyl Phosphate Metabolites* (Scorecard 2011)

Exhibit 29: National Biomonitoring Program, *Organophosphorus Insecticides: Dialkyl Phosphate Metabolites* (United States Centers for Disease Control and Prevention, 2013)

Exhibit 30: Gershwin, M.E., *et al*., *Electrophile-modified lipoic derivatives of PDC-E2 elicits anti-mitochondrial antibody reactivity* (Journal of Autoimmunity ed. 37, 2011)

Exhibit 31: Husain, K., *Delayed Neurotoxicity of Organophosphorus Compounds* (J. of Environ. Immun. and Toxicology, Vol. 1, issue 1, Mar./Apr. 2013)

Exhibit 32: Metcalf, R.I., *et al*., *Meta-sulfurpentafluorophenyl Diethyl Phosphate and Meta-sulfurpentafluorophenyl N-methylcarbamate as Insecticides and Anticholinesterases* (J. Of Economic Entomology, Vol. 55, No. 3, June 1962)

Exhibit 33: Yamashita, T., *et al., Simultaneous removal of colour, phosphorus and disinfection from treated wastewater using an agent synthesized from amorphous silica and hydrated lime* (Environ Technol., ed. 34, 2013)

Exhibit 34: Smith, K., *et al*., *New Reagent Systems for Electrophilic Chlorination of Aromatic Compounds: Organic Chlorine-Containing Compounds in the Presence of Silica* (Dept. of Chemistry, Univ. College of Swansea, U.K., Dec. 1985)

Exhibit 35: Occupational Safety and Health Association, *Occupational Exposure to Respirable Crystalline Silica – Review of Health Effects Literature and Preliminary Quantitative Risk Assessment* (Docket OSHA-2010-0034)

Exhibit 36: *Organophosphorus Insecticides: Dialkyl Phosphate Metabolites* (United States Centers for Disease Control and Prevention, 2009)

Exhibit 37: Petty, C.S., *et al*., *Organic Phosphate Insecticides – A Survey of Blood Cholinesterase Activity of Exposed Agricultural Workers in Louisiana, 1957* (Am. J. Public Health, Vol. 49, No. 1, Jan. 1957)

Exhibit 38: Blanc-Lapierre, A., *et al*., *Cognitive Disorders and Occupational Exposure to Organophosphates: Results From the PHYTONER Study* (Am. J. of Epidemiology, Vol. 177, No. 10, 2013)

Exhibit 39: Wang, H.P., *et al*., *H NMR-based metabonomic analysis of the serum and urine of rats following subchronic exposure to dichlorvos, deltamethrin, or a combination of these two pesticides* (Chemico-Biological Interactions, Vol. 203, 2013)

Exhibit 40: Marx, R., *Uncovering the Cause of "Phossy Jaw" Circa 1858 to 1906: Oral and Maxillofacial Surgery Closed Case Files—Case Closed* (Am. Assoc. of Oral and Maxillofacial Surgeons, 2008)

Exhibit 41: Sanfilippo, F., *et al*., *Crystalline Deposits of Calcium and Phosphorus* (Arch. Pathol. Lab. Med., Vol. 105, Nov. 1981)

Exhibit 42: Thedrez, A., *et al*., *IL-21-Mediated Potentiation of Antitumor Cytolytic and Proinflammatory Responses of Human Vγ9Vδ2 T Cells for Adoptive Immunotherapy* (The J. of Immunology, 2009)

Exhibit 43: Aff. of Dr. M. Eric Gershwin, dated Oct. 17, 2013

Exhibit 44: Salama, A., and Little, M., *Animal models of ANCA associated vasculitis* (Curr. Opin. Rheumatol., January 2012)

Exhibit 45: Kallenberg, C., *Pathophysiology of ANCA-Associated Small Vessel Vasculitis* (Curr. Rheumatol Rep, Vol. 12, 2010)

Exhibit 46: *Robelin v Spectrum Health Hosps*, unpub'd op *per curiam* of the Court of Appeals, issued Sept 10, 2009 (Dkt. No. 279780); 2009 Mich App LEXIS 1865

Exhibit 47: *Clerc v Chippewa County War Mem Hosp*, unpub'd op *per curiam* of the Court of Appeals, issued Nov 14, 2013 (Dkt. No. 307915); 2013 Mich App LEXIS 1823

Exhibit 48: January 29, 2015 COA Order Reversing Exclusion of Dr. Gershwin

Exhibit 49: January 29, 2015 COA Dissent Regarding Order Reversing Exclusion of Dr. Gershwin

Exhibit 50: August 16, 2016 COA Order Reversing Exclusion of Dr. Gershwin, On Remand

Exhibit 51: *Elher v Misra*, 499 Mich 11; 878 NW 2d 790 (Dkt. No. 150824, 2016)

Exhibit 52: April 25, 2016, MSC Order Vacating and Remanding re *Elher*

PROOF OF SERVICE

I hereby certify that on ***June 21, 2017***, I electronically filed ***Plaintiffs-Appellees' Supplemental Brief RE Leave to Appeal (After Remand) on Order of the Court Dated May 10, 2017*** with the Clerk of the Court using the TrueFiling file and serve system which will send notification of such filing to counsel of record.

/s/ Shannon Shaw